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Gas exchange during inhalation anaesthesia of horses: a comparison between immediate versus delayed start of intermittent positive pressure ventilation – A clinical study

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Summary

This clinical study compares the effects of an immediate versus a delayed start of IPPV on pulmonary function and gas exchange in horses anaesthetized with isoflurane. Thirty healthy warm-blood horses (twelve mares, eight geldings and ten stallions) presented for elective surgical and diagnostic procedures. Median body weight was 528 kg (range 335-650 kg) and median age 7,5 years (range 2-15 years). After pre-anaesthetic medication with romifidine anaesthesia was induced with intravenous diazepam and ketamin. Horses were intubated and positioned in lateral or dorsal recumbency. Anaesthesia was maintained with isoflurane in 100% oxygen. Horses assigned to group I (n=15) were ventilated immediately after induction. Horses assigned to Group II (n=15) were breathing spontaneously for 40 minutes before intermittent positive pressure ventilation (IPPV) was applied. Respiratory and haemodynamic variables were measured or calculated. Analyses of arterial blood gases were done 20 (T20), 40 (T40) and 55 (T55) minutes after induction of anaesthesia. PaO_2 and (A-a)PO_2 were significantly different in both groups at all three measuring points. In group I, the PaO_2 values were higher [382±65 vs. 207±131 mmHg (T20), 409±72 vs. 213±106 mmHg (T40) and 415±88 vs. 276±124 mmHg (T55)] and (A-a)PO_2 values significantly lower [176±58 vs. 298±111 mmHg (T20), 179±66 vs. 325±114 mmHg (T40) and 184±81 vs. 323±133 mmHg (T55)] compared to those of group II. The $PaCO_2$ of group I was significantly lower than in group II at all measuring points: [45±4 vs. 58±6 mmHg (T20), 45±5 vs. 63±9 mmHg (T40) and 45±5 vs. 50±5 mmHg (55 min)], respectively. The alveolar dead space fraction was larger in horses of group II compared to those in group I [0.08±0.09 vs. 0.18±0.13 (T20), 0.11±0.11 vs. 0.21±0.16 (T40) and 0.10±0.07 vs. 0.19±0.07 (55 min)].The results of this study suggest that an immediate start of IPPV reduces the degree of impairment of gas exchange when compared to a delayed start. This might be due to a better matching of ventilation and perfusion, fewer atelectasis and less right-to-left shunt.

Keywords: anaesthesia, horse, mechanical ventilation, IPPV, gas exchange, atelectasis

Gasaustausch während Inhalationsanästhesien beim Pferd: Vergleichende Untersuchung von sofortigem und verzögertem Einsatz von IPPV – Eine klinische Studie

Untersucht wurden die unterschiedlichen Effekte von sofortigem und verzögertem Einsatz von IPPV auf die Lungenfunktion und den Gasaustausch bei anästhesierten Pferden. Die Daten von 30 Warmblütern (12 Stuten, 8 Wallache und 10 Hengste) wurden ausgewertet. Das Gewicht (Median) lag bei 528 kg (335-650 kg) und das Alter (Median) bei 7,5 Jahren (2-15 Jahre). Nach Prämedikation mit Romifidin wurde die Anästhesie mit Diazepam und Ketamin eingeleitet. Die Pferde wurden intubiert und in Seiten- oder Rückenlage positioniert.Die Anästhesie wurde in beiden Gruppen mit Isofluran in nahezu 100% Sauerstoff aufrecht erhalten. 15 Tiere der Gruppe II atmeten spontan und wurden nach 40 Minuten mechanisch beatmet (IPPV). Die anderen 15 Patienten (Gruppe I) wurden sofort mit IPPV kontrolliert beatmet. Respiratorische und hämodynamische Parameter wurden kontinuierlich gemessen oder berechnet. Eine arterielle Blutgasanalyse wurde 20, 40 und 55 Minuten nach Anästhesiebeginn durchgeführt. Die Parameter für die Oxygenation PaO_2 und (A-a)PO_2 waren signifikant unterschiedlich in den Gruppen zu allen drei Messzeitpunkten. In Gruppe I lagen die PaO_2-Werte über [382±65 und 207±131 mmHg (T20), 409±72 und 213±106 mmHg (T40) und 415±88 und 276±124 mmHg (T55)] und die (A-a)PO_2-Werte deutlich unter [176±58 und 298±111 mmHg (T20), 179±66 und 325±114 mmHg (T40) und 184±81 und 323±133 mmHg (T55)] denen der Gruppe II. Der arterielle Kohlendioxidpartialdruck der Gruppe I lag zu jedem Messzeitpunkt signifikant unter dem der Gruppe II [45±4 und 58±6 mmHg (T20), 45±5 und 63±9 mmHg (T40) und 45±5 und 50±5 mmHg (T55)]. Bezüglich des berechneten alveolaren Totraumes kam es ebenfalls zu jedem Messzeitpunkt zu einem signifikanten Unterschied zwischen den Gruppen. Der Wert lag in Gruppe II immer über dem in Gruppe I [0,08±0,09 und 0,18±0,13 (T20), 0,11±0,11 und 0,21±0,16 (T40) und 0,10±0,07 und 0,19±0,07 (T55)]. Die Ergebnisse dieser Studie belegen, dass ein unverzüglicher Start der IPPV- Beatmung die Entstehung von Atelektasen und damit das intrapulmonale Rechts- Links- Shuntvolumen sowie die Totraumventilation bei Pferden in Inhalationsanästhesie reduziert.

Schlüsselwörter: Anästhesie, Pferd; künstliche Beatmung, IPPV, Gasaustausch; Atelektasen

Introduction

During general anaesthesia and recumbency horses often develop pronounced disturbances of gas exchange characterised by hypercapnia, hypoxemia and large alveolar to arterial partial pressure differences for oxygen (A-a)PO_2 (*Hall* et al. 1968, *McDonell* 1974, *Hall* 1979). The use of intermittent positive pressure ventilation (IPPV) and high inspired concentrations of oxygen allows correction of hypercapnia but cannot always prevent hypoxemia.

Computertomographic studies in ponies have demonstrated that during general anaesthesia pronounced atelectasis in dependent lung regions develops within twenty minutes. Furthermore, the amount of atelectasis correlates well with the

increase in right-to-left intrapulmonary shunt. It is thought that in adult horses atelectasis formation in dependent lung regions and increased shunt are also the cause of poor oxygenation (*Nyman* et al. 1990).

In an attempt to reduce the overall impairment of gas exchange IPPV has been used in combination with PEEP but results have been very variable with high levels of PEEP causing severe cardiovascular side effects (*Swanson* and *Muir* 1986, *Nyman* 1998, *Moens* 1994). The point in time at which spontaneous respiration is replaced by IPPV is very variable and depends mainly on the anaesthetic management of individual anaesthetists. It has been suggested that an early start of IPPV might reduce worsening of gas exchange and improve oxygenation after induction of anaesthesia (*Day* et al. 1995, *Smith* 1997)

This clinical study was designed to test the hypothesis that an immediate start of IPPV provided better gas exchange than a delayed start during inhalational anaesthesia in horses.

Material and Methods

Inclusion

The patient population for the study consisted of horses undergoing elective surgical and diagnostic procedures. Only horses considered healthy after physical examination and routine blood examination were included.

Exclusion

Horses with a body mass < 300kg, younger than 2 years or older than 15 years were excluded. Horses undergoing colic or laparoscopic surgery as well as those receiving bronchodilators were also excluded. Data from horses requiring less than 60 minutes general anaesthesia were not analyzed. Horses in group II in which IPPV had to be initiated earlier than 40 minutes after induction because of apnoe or because of a high $PaCO_2$ (> 70 mmHg) or low PaO_2 (< 70 mmHg) at the first arterial blood gas analysis were excluded from analysis.

Horses were randomly assigned to two groups. Horses of group I received IPPV immediately following intubation whereas in horses of group II IPPV was started only after 40 minutes of spontaneous ventilation. In group I 5 horses were operated in dorsal and 10 horses in lateral recumbency position whereas in group II these numbers were 7 and 8 respectively. Food was withheld during 24 hours but free access to water was allowed.

Anaesthesia

The horses were premedicated with 0.08 mg/kg i.v romifidinhydrochloride (Sedivet®). Induction of anaesthesia was done with 0.1 mg/kg diazepam (Diazepam.ratiopharm®) intravenously immediately followed by 3.0 mg/kg ketamin (Ursotamin®). Subsequently the horses were moved on the operating table and positioned in dorsal or lateral recumbency. Following endotracheal intubation the horses were connected to a large animal anaesthetic circle system featuring a large animal ventilator (Stephan Respirator GT®, Stephan GmbH Medizintechnik Quickborn, Germany). Isofluran (Isoflo®) carried in 100% oxygen was used to maintain anaesthesia. End–tidal isoflurane concentration was adapted to obtain a clinically adequate surgical depth of anaesthesia based on clinical observation of eyelid reflexes, position of the globe and changes in cardio-respiratory parameters.

In horses of Group I IPPV was always started within less than 5 minutes after intubation. Horses in group II were ventilated after 40 minutes of spontaneous ventilation. Controlled IPPV was administered in a pressure controlled mode. The pressure control level was adapted to deliver a tidal volume of 15 ml/kg BW. The respiratory rate was set at 5 breaths/min. Whenever mean arterial blood pressure (MABP) remained below 60 mmHg for ten consecutive minutes a continuous rate infusion of Dobutamin (Dobutamin Solvay®) was started at a dose of 0.5µg/kg/min and doubled if the result was unsatisfactory.

Measurements

The following parameters were monitored continuously: ECG and Heart rate (Dräger PM 8050®, Dräger Medical, Lübeck, Germany), invasive arterial blood pressure (a. facialis, Dräger PM 8014®, Dräger Medical; Druckwandler: Novatrans II MX 860®, Medex, Landshut, Germany), respiratory gas concentrations of CO_2, O_2 and isoflurane (Capnomac Ultima™ Datex-Ohmeda). Spirometric data (tidal and minute volume, I:E ratio, dynamic compliance, airway pressure and respiratory rate) were collected using a dedicated monitor module (Capnomac Ultima™ Datex-Ohmeda) and a pitot tube-based sensor (*Moens* 2009) All apparatus were calibrated according to manufacturers' instructions.

Arterial blood samples were taken 20 (T20), 40 (T40) and 55(T55) minutes after intubation and immediately analysed (ABL TM 5 Radiometer® Kopenhagen) for partial pressure of oxygen (PaO_2; mmHg) and carbon dioxide ($PaCO_2$; mmHg) and pH.

The alveolar-arterial partial pressure difference for O_2 [(A-a)PO_2] was automatically calculated by the blood gas analyzer based on the alveolar gas equation using correction for body temperature and the actual inspired fraction of oxygen (FIO_2).

The alveolar dead space fraction was calculated using following formula: $VD_{alv}/VT_{alv} = (PaCO_2\text{-}PE'\ CO_2/PaCO_2)$ where $PE'\ CO_2$ is the end- expired PCO_2 and VT alveolar tidal volume (*Moens* 1989, *Fletscher* 1990, *Hedenstierna* and *Sandhagen* 2006).

The total amount of dobutamine administered was calculated.

Statistical methods

Statistical analysis was performed using SPSS11.5. (SPSS Software GmbH, München, Germany). The distribution of data

Table 1 Mean values ± SD of tidal volume (VT), minute volume (MV), peak inspiratory pressure (PIP) and respiratory rate of group I (immediate IPPV) and II (delayed IPPV).

Variables	Group	T20	T40	T55
VT (L)	I	7.89 ± 1.08	7.94 ± 1.26	7.81 ± 1.16
	II	6.55 ± 2.35	7.08 ± 2.78	7.73 ± 1.18
MV (L)	I	38.3 ± 6.0	38.6 ± 5.7	38.7 ± 5.7
	II	36.3 ± 15.8	31.4 ± 13.0	37.1 ± 5.5
PIP (cmH_2O)	I	24.6 ± 2.2	24.5 ± 2.9	24.9 ± 2.9
	II			23.9 ± 2.7
RR (Breaths/min)	I	4.9 ± 0.3	5.0 ± 0	5.0 ± 0
	II	5.9 ± 2.4	5.1 ± 3.1	5.0 ± 0

was analysed using the SHAPIRO-WILK-Test and normally distributed data presented as mean and standard deviation. Data for age and body mass were not normally distributed and are thus shown as median and range. The influence of group assignment, time and differences between groups at T20, T40 and T55 were tested for using the LEVENE and T-tests for independent variables. Non-normally distributed data were analysed by the MANN-WHITNEY U-test. Comparison between groups for sex, body position and quantity of dobutamine were done using the Chi2-Test. A $p < 0.05$ was considered significant.

Results

There was no statistical difference between groups I and II for age or body weight. There were no significant differences between the groups in end expiratory isoflurane concentration (FE'iso) and dobutamin consumption at any time point.

In group I (n=15) PaO_2 was greater, (A-a)PO_2, $PaCO_2$ and alveolar dead space fraction lower at all points in time compared to group II (n=15). Blood gas values, alveolar dead space fraction and pH are given in table 2, figure 1 and 3.

Spirometric data and MABP (Figure 2) are given in table 1. There were no differences between the groups except for lower minute ventilation during spontaneous respiration during IPPV.

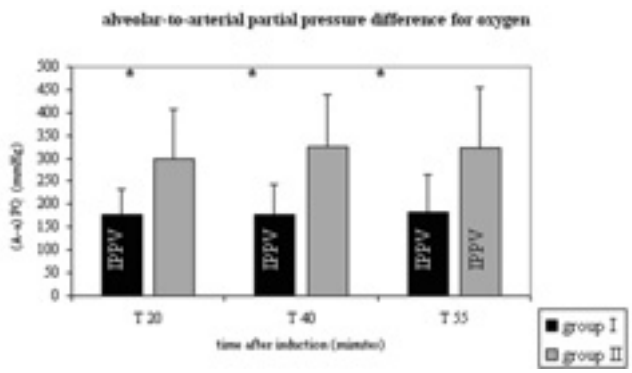

Fig. 1 Alveolar-to-arterial partial difference for Oxygen for group I (immediate IPPV) and Group II (delayed IPPV) at 20, 40 and 55minutes after induction of general anaesthesia. *: Significant difference between group I and II.

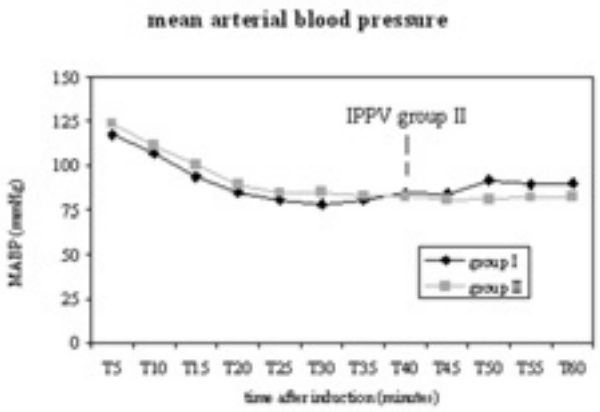

Fig. 2 Mean arterial blood pressure at different time points during general anaesthesia for group I (immediate IPPV) and Group II (delayed IPPV)

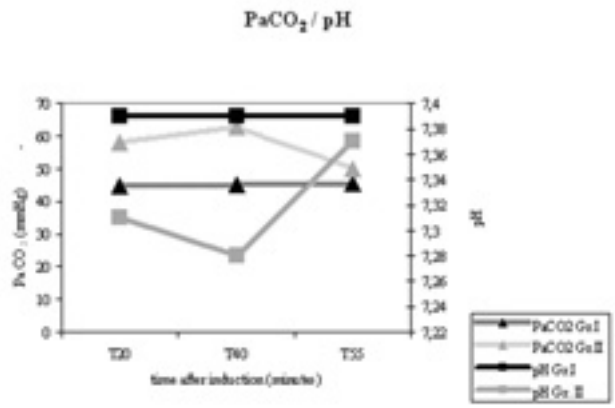

Fig. 3 Mean arterial partial pressure for CO2 (PaCO2) and pH for group I (immediate IPPV) and Group II (delayed IPPV) at 20, 40 and 55minutes after induction of general anaesthesia

Discussion

The main finding of this study was a better gas exchange in those horses, which received IPPV immediately after the induction of general anaesthesia as compared to those with a delayed start of IPPV after a period of spontaneous breathing of 40 minutes.

The management of equine anesthesia and particularly of IPPV differs among veterinary anaesthetists. Especially the

Table 2 Mean values ± SD for arterial partial pressure of oxygen (PaO2), alveolar- to- arterial partial pressure differences for oxygen (P(A-a)O2), arterial partial pressure of carbon dioxide (PaCO2), calculated alveolar dead space fraction (VDalv / VT), pH and mean arterial blood pressure of group I (immediate IPPV) and II (delayed IPPV). *: Significant difference between group I and II.

Variables	Group	T20	T40	T55
P_aO_2 (mmHg)	I	381.5 ± 65.0*	408.9 ± 71.5*	415.1 ± 88.3*
	II	206.9 ± 130.8	212.5 ± 105.7	275.7 ± 124.0
$P(A\text{-}a)O_2$ (mmHg)	I	176.0 ± 58.0*	178.5 ± 65.9*	183.7 ± 81.2*
	II	297.7 ± 110.6	324.9 ± 113.6	322.5 ± 133.3
P_aCO_2 (mmHg)	I	44.6 ± 4.2*	45.1 ± 4.9*	45.5 ± 5.4*
	II	58.1 ± 6.1	62.7 ± 8.9	49.9 ± 5.1
VD_{alv} / VT_{alv}	I	0.08 ± 0.09*	0.11 ± 0.11*	0.10 ± 0.07*
	II	0.18 ± 0.13	0.21 ± 0.16	0.19 ± 0.07
pH	I	7.39 ± 0.03*	7.39 ± 0.04*	7.39 ± 0.04
	II	7.31 ± 0.05	7.28 ± 0.05	7.37 ± 0.05
MABP	I	81.4 ± 22.0	81.9 ± 19.4	81.9 ± 19.4
	II	92.3 ± 28.7	84.7 ± 21.3	84.7 ± 21.3

decision whether or not to use IPPV and if so, when to start mechanical ventilation differs among individuals. Some anaesthetists will start IPPV only when hypoxemia of severe hypercapnia are suspected or have been confirmed during spontaneous respiration, whereas other anaesthetists will use IPPV routinely from the very beginning of anaesthesia regardless of the efficiency of gas exchange at that moment.

The order of magnitude of the derangement of gas exchange found in the current study is in line with what has been reported in the literature (*Hall* et al. 1968, *Nyman* et al. 1990, *Day* et al. 1995). As expected, horses in group II suffered from hypercapnia, hypoxia and an increased $(A\text{-}a)PO_2$ during spontaneous breathing. The installation of IPPV after 40 minutes reversed the state of hypoventilation and normalized $PaCO_2$ but was unable to correct hypoxemia with PaO_2 values remaining essentially unchanged. This limited effect of IPPV is in line with numerous reports in the literature (*Day* et al. 1995, *Nyman* and *Hedenstierna* 1989, *Beadle* et al 1975).

The lower arterial pH in group II reflects mainly the development of respiratory acidosis accompanying hypoventilation and increased $PaCO_2$ during spontaneous respiration. However pH increased and was not different anymore from Group I when IPPV was used in group II and $PaCO_2$ decreased towards normal values

Horses in group I were essentially normocapnic during the entire procedure despite an increased $(A\text{-}a)PO_2$. However, their efficiency of gas exchange was significantly better than in group II at T20 and T40 (spontaneous respiration in group II) and T55 (IPPV in group II).

In the presence of high FIO_2 $(A\text{-}a)PO_2$ differences were increased in both groups indicating right-to-left shunting. It is thought that in anaesthetized horses atelectasis formation in dependent lung regions is at the origin of this shunt. Hence the results in group I of this study suggest that the immediate start of IPPV could reduce but not prevent atelectasis formation and shunting. Furthermore, it is suggested that atelectasis and shunts were more pronounced in group II and not influenced by the start of IPPV.

Alveolar collapse in dependent lung regions is caused by a direct compression of lung tissue and by absorption of oxygen from alveoli in poorly ventilated lung units. As compared to smaller species gravity-dependent compressive forces are far more pronounced in horses. When horses are placed in the recumbent position the dome shaped diaphragm and the voluminous abdominal organs exert an additional compression. It is unlikely that the intermittent rise in airway pressure during IPPV can counteract these permanent compressive forces. Therefore, it is more likely that IPPV increases ventilation in the poorly ventilated alveolar units instead of actively opening collapsed lung units.

There is ample evidence that the disturbances in gas exchange develop in the horse within half an hour following induction of general anaesthesia. Computertomographic studies in humans, sheep and ponies have demonstrated that atelectatic regions are well established in dependent lung regions after 20 minutes (*Nyman* et al. 1990, *Rothen* et al. 1995, 1999).

The exact interval between induction of anaesthesia and the start of IPPV is seldom reported. In a study comparing spontaneous respiration and IPPV in 160 horses (*Day* et al. 1995) started IPPV after 20 minutes. At this moment gas exchange had already declined and was considered to be due to the formation of atelectasis. These authors suggest that an earlier start of IPPV might have prevented or at least reduced alveolar collapse. *Smith* et al. (1997) found a higher PaO_2 in colic horses when IPPV was started within 10 minutes compared to 15-60 minutes after induction.

The study presented here confirms the ineffectiveness of IPPV to improve $(A\text{-}a)PO_2$ and oxygenation after a period of spontaneous respiration, which has been documented over the past decades (*Nyman* et al. 1987, *Tokics* et al.1987, *Teixeira* et al. 2000). These negative results suggest that classical IPPV with peak inspiratory pressures of <30 cm H_20 and tidal volumes up to 15 ml/kg does not reopen collapsed alveoli to make them available for gas exchange. In human anaesthesia and intensive care medicine specific ventilation strategies aimed at reopen collapsed lung units have received much attention in the recent past (*Tusman* et al. 1999, 2004). The-

se strategies use "alveolar recruitment manoeuvers" which apply specific sequences of high peak inspiratory pressures (PIP) and PEEP to open up collapsed alveoli. *Lachmann* (1992) was the first to proclaim that high PIP should be used to open collapsed alveoli while sufficient PEEP was to be used to keep the newly recruited alveoli open. The most prominent derivates of his proposal used in clinical practice are the "open lung approach" by *Amato* (1995) and the "alveolar recruitment strategy" by *Tusman* et al. (1999, 2004). The pulmonary patho-physiology, seen in anaesthetized morbidly obese humans also features as is the case in equidae pronounced atelectasis formation (*Rothen* et al. 1995). The effectiveness of the use of PEEP and alveolar recruitment manoeuvers has been studied in this patient category. It has been demonstrated that the mere addition of PEEP to IPPV without previous alveolar recruitment in attempt to improve oxygenation has yielded similar limited results as documented in horses (*Almarakbi* et al 2009, *Bohm* et al. 2009, *Reinius* et al 2009). The feasibility and efficacy of such alveolar recruitment maneuvers in equidae are currently under investigation (*Wettstein* et al. 2006, *Levionnois* et al. 2006, *Iff* 2007, *Schürmann* 2008).

IPPV settings in the present study were identical in both groups and measured minute volume during IPPV was not different. However, alveolar dead space fractions (VD_{alv}/VT_{alv}) in group II were larger at all times. This is possibly due to a shift of ventilation towards relatively overinflated alveoli in group II as overall ventilation has to be achieved in a smaller aerated total lung volume. The larger alveolar dead space fraction at similar minute ventilation can explain the higher $PaCO_2$ in group II. The lower $PaCO_2$ and better oxygenation seen in group I suggest that ventilation and perfusion are better matched when IPPV is started right away.

The increase in mean intra-thoracic pressure that accompanies IPPV may cause a variable degree of cardiovascular depression due to a decrease in venous return to the heart. These negative hemodynamic effects can become even more pronounced when IPPV is superimposed on the transitory circulatory side effects of short acting induction drugs. The change from a standing to a recumbent position might exert additional effects on hemodynamics that are unpredictable in magnitude and direction. In the current study there were no differences in MABP and in the total amount of inotropic support at any point in time between the two groups (table 2, figure 2). Although cardiac output was not measured in this study population of healthy horses the surrogate hemodynamic parameters suggest that the circulatory side effects of anaesthesia induction where influenced very little by the immediate institution of IPPV.

In conclusion the results of this study suggest that an immediate in stead of a delayed start of IPPV after induction of anaesthesia can reduce the impairment of gas exchange typically seen in anaesthetized horses.

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References

Amato M. B., Barbas C. S., Medeiros D. M., Schettino G. P , Lorenzi Filho G., Kairalla R. A., Deheinzelin D., Morais C., Fernandes E. O, Takagaki T. Y. et al. (1995) Beneficial effects of the ''open lung approach'' with low distending pressures in acute respiratory distress syndrome. A prospective randomized study on mechanical ventilation. Am. J. Respir. Crit. Care Med. 152:1835–1846

Almarakbi W. A., Fawzi H. M. and *Alhashemi J. A.* (2009) Effects of four intraoperative ventilatory strategies on respiratory compliance and gas exchange during laparoscopic gastric banding in obese patients. Br. J. Anaesth. 102, 862-868

Beadle R. E., Robinson N. E. and *Sorenson P. R.* (1975) Cardiopulmonary effects of positive end expiratory pressure in anesthetized horses. Am. J. Vet. Res. 36, 1435-1438

Böhm S. H., Thamm O. C., von Sandersleben A., Bangert K., Langwieler T. E., Tusman G , Strate T. G. and *Standl T. G.* (2009) Alveolar recruitment strategy and high positive end-expiratory pressure levels do not affect hemodynamics in morbidly obese intravascular volume-loaded patients. Anesth. Analg. 109, 160-163

Day T. K., Gaynor J. S., Muir W. W., Bednarski R. M. and *Mason D. E.* (1995) Blood gas values during intermittent positive pressure ventilation and spontaneous ventilation in 160 anesthetized horses positioned in lateral or dorsal recumbency. Vet. Surgery 24, 76-266

Fletcher R. (1990) Deadspace during anaesthesia. Acta Anaesthesiol. Scand. 34 (Suppl 94) 46-50

Hall L. W. (1979) Oxygenation of Pulmonary Vein Blood in conscious an anaesthetised Ponies. Equine vet. J. 11, 71-75

Hall L. W., Gillespie J. R. and *Tyler W. S.* (1968): Alveolar- arterial oxygen tension differences in anaesthetised horses. Br. J. Anaesth. 40, 560-568

Hedenstierna G. and *Sandhagen B.* (2006) Assessing dead space. A meaningful variable? Minerva Anesthesiol. 72, 521-528

Iff I., Levionnois O. and *Moens Y.* (2007) Alveolar recruitment manoeuvre in a Shetland pony anesthetized for laparcopic castration. Wien. Tierärztl. Wschr. 94, 11-12, 264-268

Lachmann B. (1992) Open up the Lung and keep the lung open. Intensive Care Med. 118, 319-321

Levionnois O., Iff I. and *Moens Y.* (2006) Successful treatment of hypoxemia by an alveolar recruitment maneuver in a horse during general anaesthesia for colic surgery. Pferdeheilkunde 22, 1-3

McDonell W. N. (1974) The effects of anaesthesia on pulmonary gas exchange and arterial oxygenation in the horse. PhD Thesis, University of Cambridge

Moens Y. (1989) Arterial-alveolar carbon dioxide tension difference and alveolar dead space in halothane anaesthetised horses. Equine Vet. J. 21, 282-284

Moens Y., Gootjes P., Jonita J-C., Heinonen E. and *Schatzmann U.* (2009) In vitro validation of a Pitot-based flow meter for the measurement of respiratory volume and flow in large animal anaesthesia. Vet. Anaesth. Analg. 36, 209-219

Nyman G., Frostell C., Hedenstierna G., Funkquist B., Kvart C. and *Blomqvist H.*(1987) Selectiv mechanical ventilation of dependent lung regions in the anaesthetized horse in dorsal recumbency. Br J. Anaesth. 59,1027-1034

Nyman G. and *Hedenstierna G.* (1989) Ventilation perfusion relationship in the anaesthetised horse. Equine Vet. J. 21, 274-281

Nyman G., Funkquist B., Kvart C., Frostell C., Tokics L., Strandberg Ä., Lundquist H., Lundh B., Brismar B. and *Hedenstierna G.* (1990) Atelectasis causes gas exchange impairment in the anaesthetised horse. Equine Vet. J. 22, 317-324

Reinius H., Jonsson L., Gustafsson S., Sundbom M., Duvernoy O., Pelosi P., Hedenstierna G. and *Fredén F.* (2009) Prevention of atelectasis in morbidly obese patients during general anesthesia and paralysis: a computerized tomography study. Anesthesiology, 111, 979-87

Rothen H. U., Sporre B. and *Engberg G.* (1995) Prevention of atelectasis during general anaesthesia. Lancet 345, 1387-1391

Rothen H. U., Neumann P., Berglund J. E., Valtysson J., Magnussen A. and *Hedenstierna G.* (1999) Dynamics of re-expansion of atelectasis during general anaesthesia. Br. J. Anaesth. 82, 551-556

Schürmann P. (2008) Optimierung des pulmonalen Gasaustausches während der Pferdenarkose durch Beatmung nach dem „Open Lung Concept". Pferdeheilkunde, 236-242

Smith M. (1997) The necessity of starting ventilation in horses from beginning of anaesthesia. Proceedings of the 6th World Congress of Veterinary Anaesthesia, Thessaloniki, Greece, p 129

Teixeira Neto F. J., Luna S. P. L., Massone F., Thomassian A., Vargas J. L. R., Junior J. R. S. et al. (2000) The effect of changing the mode of ventilation on the arterial-to-end-tidal CO_2 difference and physiological dead space in laterally and dorsally recumbent horses during halothane anesthesia. Vet. Surg. 29, 200-205

Tokics L., Hedenstierna G., Strandberg A., Brismar B. and *Lundquist H.* (1987) Lung collapse and gas exchange during general anesthesia: effects of spontaneous breathing, muscle paralysis, and positive end-expiratory pressure. Anesthesiology 66, 157-67

Tusman G., Böhm S. H., Vazquez De Anda G. F., Do Campo J. L. and *Lachmann B.* (1999) Alveolar recruitment strategy improves arterial oxygenation during general anaesthesia. Br. J. Anaesth. 82, 8-13

Tusman G., Böhm S. H., Suarez- Sipmann F. and *Turchetto E.* (2004) Alveolar recruitment improves ventilatory efficiency of the lungs during anesthesia. Can. J. Anesth. 51, 723-727

Wettstein D., Moens Y., Jaeggin-Schmucker N., Böhm S. H., Rothen U. R., Mosing M., Kästner S. B. R. and *Schatzmann U.* (2006) Effects of an alveolar recruitment maneuver on cardiovascular and respiratory parameters during total intravenous anesthesia in ponies. Am. J. Vet. Res. 67, 152-159

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